SPONTANEOUS PULMONIC INTERSTITIAL AND MEDIASTINAL EMPHYSEMA IN AN INFANT*

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ALTHOUGH interstitial emphysema of the lungs and mediastinum probably occurs more frequently than is at present recognized, as Hamman's recent paper[1] indicates, it seldom produces a fatal outcome. Fisher and Macklin[2] have reported a fatal case of this condition in a young child in whom the initial cause was the aspiration of peanut fragments into the right main bronchus. In their paper the relevant literature has been included up to January, 1939. Subsequent to that date little has appeared in the literature concerning this subject. McMann and Purcell[3] have described a case of subcutaneous emphysema in a newborn infant in which a pneumothorax and mediastinal emphysema were present, presumably due to sudden increase in intrapulmonary pressure during vigorous respiratory efforts, thus differing from the case here reported in which the emphysema occurred spontaneously. Moreover, their case recovered, the emphysema completely disappearing in five days, whereas in my case the infant died in a few hours. Instead of direct leakage of air into the pleural cavity from the alveoli through a rupture in the pleura, would it not be more reasonable to conclude that the air followed the pathway to the mediastinum and pleural cavity, as described by Macklin?[4] Slot and Brown[5] have reported a case similar to that of McMann and Purcell. McGuire and Bean[6] observed clinically a case of "spontaneous" pulmonic interstitial emphysema presenting a syndrome similar to that described by Hamman and an additional instance of this condition occurring in a patient following labour. Both patients made uneventful recoveries. Recently, Marcotte, Phillips, Adams and Livingstone[7] have pointed out the hazards accompanying the use of positive pressure intratracheal anæsthesia. Such complications, too often terminating fatally, are mediastinal emphysema and pneumothorax.

In this paper it is desired to record the findings in a case of spontaneous pulmonic interstitial and mediastinal emphysema occurring in a newborn infant, which terminated fatally in a few hours.

CASE REPORT

Baby P., a full term, white, female infant, weighing 7 pounds 15 ounces, was born at 2.45 a.m. on October 18, 1938. The mother was a gravida II, aged 24. Her first pregnancy resulted in the normal spontaneous delivery of a normal, living infant, weighing 7 pounds 8 ounces. The antenatal course in this second pregnancy was uneventful, although she was under treatment for syphilis. Her blood serum gave negative Kahn and Hinton tests.

Delivery was normal and spontaneous. No difficulty in resuscitation was encountered. The infant's colour was normal. A routine mucus tube (rubber catheter) was used to clear the throat and mouth. Nothing unusual about the infant was noticed at birth. At no time was there strenuous gasping or unusually vigorous crying. About 3½ hours after birth it was noticed that the baby was very quiet and not crying. Blood-tinged froth was exuding from the mouth and she was quite cyanosed. Auscultation of the chest is not recorded. Oxygen was administered under low pressure by holding a funnel over the nose and mouth a short distance from the face. The cyanosis did not improve, gradually increasing in intensity, becoming generalized and accompanied by dyspnœa. The baby died 5 hours and 45 minutes after birth.

Autopsy.—An autopsy was performed one hour after death. Before opening the thorax pneumothorax was tested for but none found.

Upon opening the thorax, attention was immediately drawn to the voluminous, over-inflated right lung and the presence of numerous gas bubbles and emphysematous blebs under the pleura at the root of both lungs, extending into the anterior and superior mediastinum (Fig. 1). A collection of rather large gas bubbles was found on the antero-medial aspect of the left lobe of the thymus. Although especially looked for, no emphysema of the neck or retroperitoneal tissues was detected. Therefore, the emphysema was confined to the lungs and mediastinum. The thoracic organs were removed *en masse* in order to study the distribution of the escaped air. The lungs were fixed in the distended state by running Bouin's fluid into the trachea under low pressure. The trachea was then ligated and the lungs and other thoracic organs were immersed in Bouin's fluid until thoroughly fixed.

The lungs were separated from their neighbouring organs by cutting through their roots. The lung tissue was cut into moderately thick sagittal slices with a thin, sharp knife. In the right lung several smooth-lined, cyst-like, subpleural blebs (gas bubbles) were seen. These varied in size from 1 mm. or less to 1.5 cm. in diameter. The lung tissue in general was moderately well inflated, containing some emphysematous blebs, but patchy, atelectatic areas were also noted. Much more striking and significant were the accumulations of gas bubbles in the perivascular sheath of the pulmonary vessels. There, the escaped air had dissected the perivascular connective tissue leaving it split into delicate, glistening, cobweb-like strands

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attached to the pulmonary vessels (Fig. 3). The latter, surrounded by gas bubbles, were markedly compressed and collapsed. This intersititial emphysema was more pronounced about the larger pulmonary vessels as they passed from the central part of the lung towards the hilus, but small vessels were similarly involved in some instances.

The chief gross pathological features are illustrated in Figs. 2 and 3. The bronchi gaped open and contained no gross exudate and no evidence of bronchial obstruction was found. The left lung in general was atelectatic and much smaller in volume than the right. Parts of the left lung sank in water and the left lung as a whole, did not float nearly as readily as did the right lung. However, some patchy areas of gross alveolar ectasia were present and a few subpleural emphysematous blebs were detected along the cardiac impression and at the hilus, but none elsewhere. The interstitial emphysema was less extensive than in the right lung. However, it was seen that, surrounding at least four pulmonary vessels, the perivascular sheath was stripped up in a similar manner to that described in the case of the right lung. The perivascular air bubbles were found chiefly in the central and hilar parts of the lung. No gross evidence of bronchial exudate or obstruction was found.

The heart showed no congenital anomaly and, apart from slight dilatation of the right atrium and right ventricle, it appeared normal. Gas bubbles were present in the interlobular connective tissue of the thymus but the thymic tissue itself showed nothing unusual. The other organs, including the brain and its coverings, presented nothing of significance.

Microscopic findings.—Microscopic sections served to confirm the gross impressions. In the left lung the alveoli for the most part were collapsed and quite

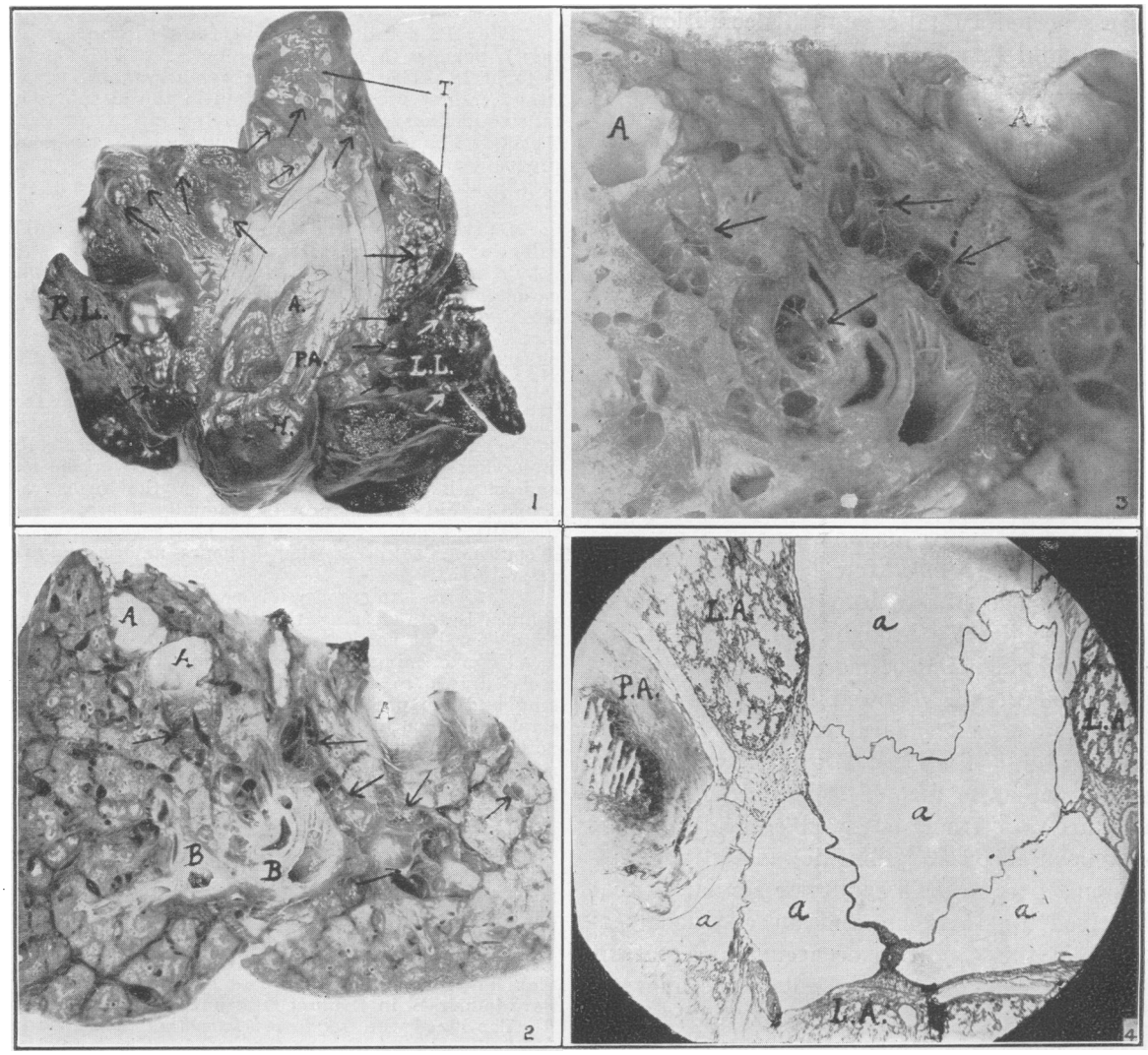

Fig. 1.—Fresh thoracic organs removed from the body *en masse* viewed from the front. The pericardium has been opened. It and the thymus have been lifted up and turned back to expose the lung roots. Air blebs (arrows) are seen over the thymus, at the lung roots and in the lung tissue. A. = aorta; P.A. = pulmonary artery; H. = heart; R.L. = right lung; L.L. = left lung; T. = thymus. **Fig. 2.**—Enlarged view of slice of fixed right lung showing subpleural air blebs (A.) and air bubbles distending and dissecting the perivascular sheaths (arrows). Note the pale, over-inflated lung tissue. B. = bronchus. x 2.2. **Fig. 3.**—Higher magnification of a field in the slice of lung shown in Fig. 2. At the top two large, subpleural blebs are seen (A.). In the central area, the perivascular sheaths have been distended with air bubbles and stripped up into delicate, cobweb-like structures (arrows). The pulmonary vessels are compressed and flattened. x 4. **Fig. 4.**—Photomicrograph of a field from the left lung showing a pulmonary artery (P.A.) on the left and air bubbles (a.) distending the perivascular sheath. Lung alveoli (L.A.) are seen surrounding the sheath. x 72.

markedly atelectatic. About some of the larger pulmonary vessels the sheaths were distended with air. The outstanding feature in the sections of the right lung was the large amount of air in the perivascular sheaths. The pulmonary vessels, both large and small ramifications, were compressed and flattened by the surrounding air pressure to such an extent as to seriously impede blood flow through them. The interstitial emphysema is illustrated in Fig. 4. The lung tissue of the right lung showed much more alveolar ectasia than that of the left lung.

COMMENT

It is probable that this infant's left lung did not expand properly after birth and remained atelectatic, being greatly diminished in volume. Consequently, an adaptive or compensatory over-inflation of the right lung, as well as localized, patchy areas of alveolar ectasia in the left lung, developed. This pronounced stretching of the alveoli probably produced multiple ruptures of the alveolar walls, allowing air to escape into the perivascular sheaths, from there to the mediastinum, and beyond in a manner fully described by Macklin.[4, 8] I am convinced that the compression of the pulmonary vessels by the air in the perivascular sheaths, together with the mediastinal emphysema, explains the infant's cyanosis, dyspnœa and circulatory failure.

Apparently healthy alveoli may rupture spontaneously without increased intrapulmonary pressure, as Hamman has pointed out. In this infant no vigorous crying occurred and no forceful methods of resuscitation were employed. I feel that the administration of oxygen had nothing to do with the development of the emphysema since the cyanosis and dyspnœa made their appearance before the administration and, further, the gas was administered through normal inhalation without any positive intratracheal pressure.

It is quite possible that the pulmonic interstitial and mediastinal emphysema in this newborn infant bore a graver significance than in an older child or adult, since in the infant the pulmonary vessels would be more compressible and the atria of the heart more collapsible than these structures in older persons, which possess a heavier, better developed musculature. In other words, it would appear that the prognosis is less favourable in the very young.

In the study of post-mortem material for pulmonic interstitial emphysema, the importance of fixing the lungs in the distended state after the method above-described is emphasized. In this way shrinkage and collapse of the lung tissue is prevented and thus dispersion of the gas bubbles and destruction of their relations to the pulmonary vessels and other lung structures are avoided.

SUMMARY

A case of pulmonic interstitial and mediastinal emphysema occurring spontaneously in a newborn infant and terminating fatally in a few hours is described. It is considered that the emphysema resulted from an over-inflation of the alveoli producing multiple ruptures and escape of air into the perivascular sheaths. It is believed that death was due chiefly to obstruction to the pulmonary and mediastinal circulation resulting in a severe degree of anoxæmia and circulatory embarrassment. While the non-fatal, clinical form of this emphysema seems to be not at all rare, it often passes unrecognized; some cases, however, occasionally end fatally. Fixation of the lungs in the distended position is important in studying the distribution of the escaped air and its relations to the lung structures.

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